THE IRREGULAR PULSE

ITS VALUE IN PROGNOSIS

STOKES-ADAMS DISEASE

REPORT OF A CASE

BY

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(4)

with [illegible]'s
Compliments.

(5) No determination of the viscosity of the blood is complete without making at the same time a careful blood-count and observing the temperature.

(6) In these estimations a good assistant will be invaluable, whose care it should be to time the rate of flow.

This instrument has been made for us by Mr Hawksley, of Oxford Street, who will supply a complete outfit in case; or, if desired, the tubes may be obtained separately.

THE IRREGULAR PULSE: ITS VALUE IN PROGNOSIS.

By JOHN HAY, M.D., M.R.C.P.,
Physician to the Stanley Hospital, Liverpool; Physician to the Hospital for Consumption and Diseases of the Chest.

IRREGULAR action of the heart is a subject covering such a wide area that I have decided to confine myself in this communication to one particular form—the commonest—known by the technical term of extra or premature systole.

In analysing the various forms of irregular action of the heart, I make use of Mackenzie's modification of Dudgeon's sphygmograph, and find it portable and fairly easy to work. By it are recorded simultaneous tracings of the jugular and radial pulses, and from these tracings one can state with a fair degree of certainty what the right and left sides of the heart are doing at any given moment.

Fig. 1 is an example of tracings obtained from a healthy adult. It shows the normal or auricular type of venous pulse. The portion E of the tracing, included between the vertical lines, indicates that period of ventricular systole during which the semilunar valves are open. The wave *a* preceding the period E can only be due to the systole of the right auricle. The wave *c* is caused by the impact of the carotid pulse.

This explanation is verified in the tracings fig. 2, which

were obtained with the metal receiver pressed more firmly on the neck. As a result of this increased pressure, the carotid pulse is dominant in the upper tracing, and the sphygmogram is typically arterial in character. The wave *a*, representing the systole of the auricle, still persists, and bears the same relationship to the carotid pulse as in fig. 1; though, as a result of the increased pressure, it is much diminished in amplitude.

We know that the muscular contraction during cardiac

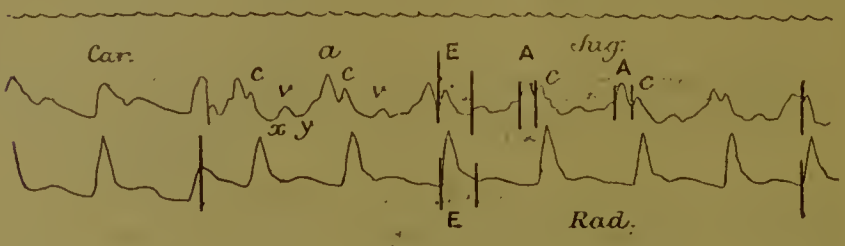

FIG. 1.

Tracing to show the normal or auricular type of venous pulse. The space E indicates in all the tracings the period of ventricular systole during which the semilunar valves are open, the sphygmic period. The wave *a*, preceding the period E, can only be due to the systole of the right auricle. The fall *x* is partly due to the diastole of the auricle causing a rush of blood out of the jugular vein into the auricle, and partly to the change in position of the auriculo-ventricular groove caused by the ventricular systole.[1] [2] The wave *c* is due to the impact of the carotid pulse. The space A represents the *a c* interval and indicates the time taken by the stimulus to pass from the auricle to the ventricle. It is normally $\frac{1}{5}$ of a second. The time marker records $\frac{1}{5}$ of a second. This applies to all the tracings.

systole spreads like a wave from the sinus of the auricles over the auricles down on to the ventricles. This is the normal sequence—auricles first, then ventricles; but it is important to note that this sequence is not invariably followed. The auricles and ventricles sometimes contract simultaneously, or the ventricle may even contract before the auricle.

Gaskell[3] has pointed out, in his classical research on the contraction of the cardiac muscle, that the heart muscle

[1] H. I. Douma, *De Analyse van het phlebogram*.

[2] A. Keith, Hunterian Lectures, *Lancet*, 1904.

[3] W. H. Gaskell, "The Contraction of Cardiac Muscle," *Schafer's Text-book of Physiology*.

possesses five properties:—Excitability—the power to respond to a stimulus; conductivity—the power to conduct that stimulus from one point to another; tonicity; contractility; and, lastly, rhythmicity.

This latter function is the one which mostly concerns us to-night. By rhythmicity we mean the power of the heart to produce rhythmically its own stimulus, independently altogether of the nervous system. The portions of the heart in which this power is most developed are chiefly the junction of the large veins with the auricles, to a less degree the junction of the auricles and ventricles, and the conus of each ventricle. We know that as a rule the stimulus arises where the large

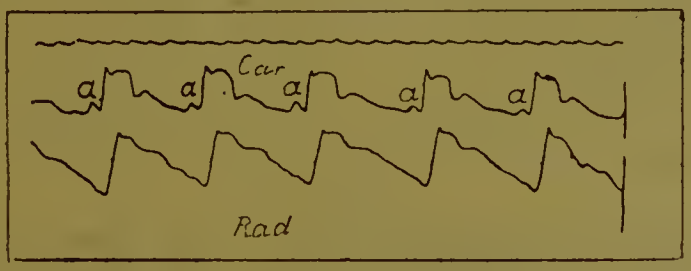

Fig. 2.

The upper tracing is from the neck, and shows a small wave (*a*) from the jugular vein and due to the systole of the right auricle preceding the large carotid wave.

veins open into the auricles; but we must not forget that every portion of the heart muscle possesses in some degree the power to initiate a contraction by the production of a stimulus.

It is the irregular action of the heart due to its response to abnormal stimuli which I wish to consider.

Your hand is on your patient's pulse; the wave is regular in force and frequency. As you keep your finger on the wrist, you notice that a beat is missed—the pulse intermits, but its rhythm is not interfered with; the dropping of the beat has not spoilt your counting. Or instead of there being a complete pause, you are conscious of a small pulse wave which occurs earlier than you anticipated, and this is followed by a long

pause, then the usual rhythm is resumed (see fig. 3). Such intermissions may occur infrequently, or they may be extremely numerous.

What are the subjective sensations of the patient in whom this irregular action of the heart occurs? In some instances we find that they are *nil*. The patient is totally unaware of anything unusual.

On the other hand, patients may tell us that when this intermission occurs they are conscious of a thud in the region of the heart, or somewhere within the chest, possibly even in

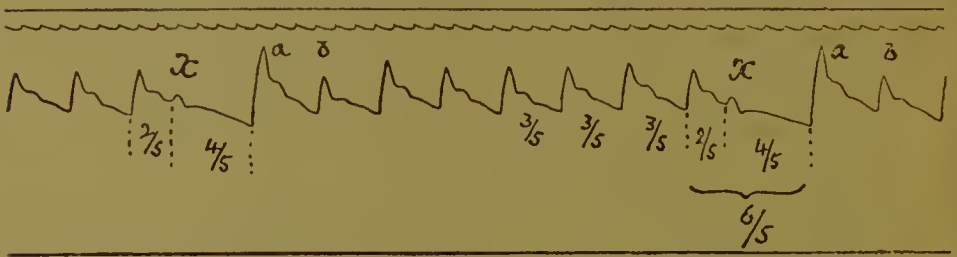

Fig. 3.

Tracing of the radial pulse showing the occurrence of two extra-systoles. Note that the normal interval is $\frac{3}{5}$ of a second, that the extra-systole occurs $\frac{1}{5}$ of a second too soon, and that the compensatory pause is $\frac{4}{5}$ of a second.

Note also that the wave *a* is larger than usual because the previous diastole has been longer, and also because the ventricle contained a larger quantity of blood when it contracted.

Note also that the wave *b* is smaller than normal, the contractility not being fully restored after the unusually vigorous systole *a*. Probably also the aortic blood-pressure was higher than normal owing to the same cause.

the neck. They may feel as if the heart stopped; or they may experience a transient sensation of giddiness, or tell you that they feel as if the heart had "rolled over"; or there may be an unpleasant sinking sensation in the cardiac region—"all-gone sensations."

It is worthy of note that, when the intermission is a constant and habitual phenomenon, the patient is far less likely to complain of any abnormal sensation than when the irregularity is transient and due to some temporary cause.

Such are the feelings of the patient.

What do we find on examining the chest? On laying the

hand over the heart we perceive an irregularity which makes itself evident by the presence of a short impulse occurring very early in the diastole, then a long pause ending in an extremely strong and forcible apex beat, followed by the normal rhythm. On auscultation one finds the same sequence.

In fig. 4 we have a diagram showing the occurrence of two extra systoles. The upper tracing is a cardiogram, and recorded simultaneously with the radial pulse.

The first extra-systole occurs so early in diastole that the

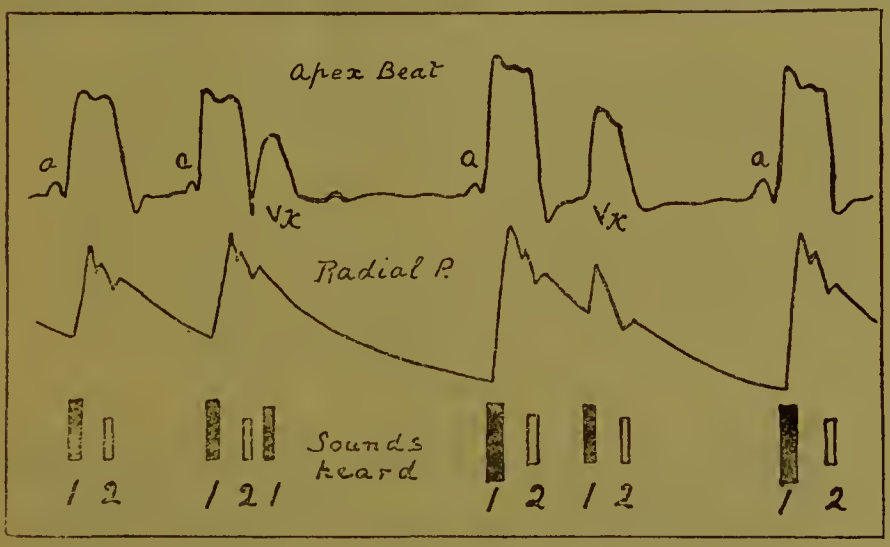

Fig. 4.

Simultaneous tracings of the apex beat and of the radial pulse, showing the occurrence of two extra systoles, *v x*. The first occurs very early in the diastole of the ventricle, the second is later in its appearance.

contraction of the left ventricle is unable to overcome the pressure in the aorta, the semilunar valves remain closed, and there is a dropped beat at the wrist. The sounds heard in such a case are represented at the foot of the diagram; and since during the first extra-systole the aortic valves remain closed, the contraction of the ventricle gives rise to a single sound; it corresponds to a first sound, and follows rapidly on the previous short, sharp second sound. Sometimes this sequence of sounds simulates very closely a reduplication of the second sound.

The second extra-systole in the diagram does not occur so early in diastole, thus allowing the ventricle a longer period of rest, in consequence of which its contractility is more fully restored, and it contains a larger quantity of blood to contract on; in addition the blood-pressure in the aorta will have fallen a little. The result of these factors is to make the extra-systole more efficient, the aortic valves are opened, blood is forced into the aorta, and a pulse can be felt at the wrist. The sounds caused by this extra-systole are two—a first sound occurring earlier in diastole than normal, and a second following

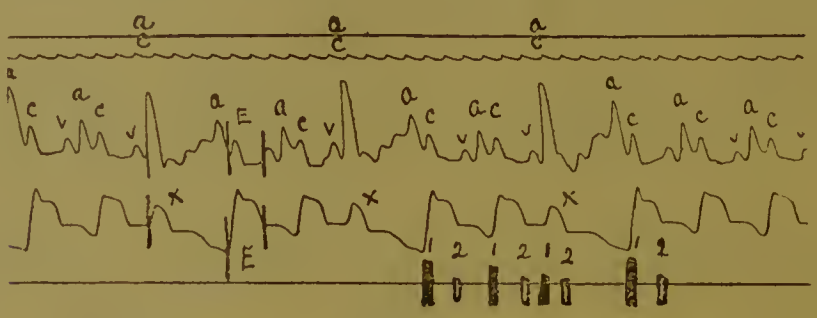

FIG. 5.

Tracings from the jugular vein and the radial artery showing a typical trigeminal pulse caused by the regular occurrence of an extra-systole; *x* marks the extra-systoles.

The jugular tracing shows that when the ventricles contract earlier than usual, as at *x*, then their systole coincides with that of the auricles, and consequently there is a large wave in the jugular vein.

a = wave due to systole of auricle. *c* = carotid pulse. E = sphygmic period.

In the markings correct allowance has been made for the delay in the radial pulse.

rapidly on the first; this is followed by a long pause terminated by a loud thumping first sound—the commencement of a normal heart-beat.

We have noted up to this point what changes are felt in the pulse, what sensations, if any, the patient experiences, and what is to be made out on examining the chest during this form of cardiac irregularity. Let us now consider to what extent an analysis of the venous pulse will assist us.

Fig. 5 shows the jugular and radial pulses during a series of extra-systoles. Every third systole being an extra-systole,

the pulse is "trigeminal." To the right of the tracing both radial and jugular pulses are normal; to the left there are three groups of the trigeminal pulse. Wherever an extra-systole occurs there is a large and noticeable wave in the jugular pulse tracing; it suddenly rises high above the average level. The reason for this high wave is that the auricles and the ventricles are contracting simultaneously. The right auricle, on contracting, finds that the auriculo-ventricular opening is closed owing to the systole of the right ventricle; in consequence of this the blood is poured back into the jugular veins. There is no other course for it.

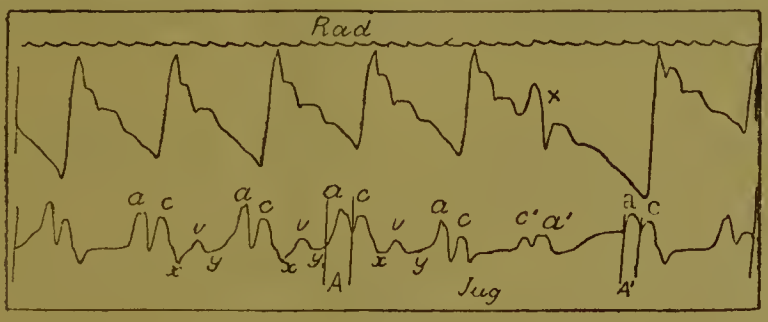

FIG. 6.

"Simultaneous tracings of the radial and jugular pulses, showing the occurrence of a ventricular extra-systole at x in the radial tracing; c' corresponds in time with x, and is due to the pulse in the carotid. The auricle continues at its regular rhythm, the wave a' occurring at its normal time, and after the ventricular systole which produced c'."

This is clearly demonstrated by the first extra-systole in the tracing, where it is evident that the period of ventricular systole, as indicated by the radial tracing, corresponds in point of time with that portion of the jugular tracing produced by the systole of the auricle.

Sometimes, as shown in fig. 6, the extra-systole of the ventricle occurs even before the contraction of the auricle; in such a case the auricular systole produces a small wave, a', in the tracing, subsequent to the jerk of the lever due to the impact of the carotid pulse, c'. In these two cases it is obvious that the ventricles contracted spontaneously and independently of the normal stimulus spreading down to them

from the auricles, for in fig. 5 we can see that the auricles and ventricles contracted simultaneously, while in fig. 6 the ventricles contracted before the auricle had initiated the normal stimulus.

In such cases there must have been some local stimulus acting on the musculature of the ventricles, causing them to contract spontaneously and independently of the auricles. These extra-systoles may appear rhythmically; every other beat thus giving rise to the so-called bigeminal pulse, or every third beat, and so producing the trigeminal pulse (see figs. 5 and 7); or these extra-systoles may happen in groups of two, three, four, or more in number, as a result of which

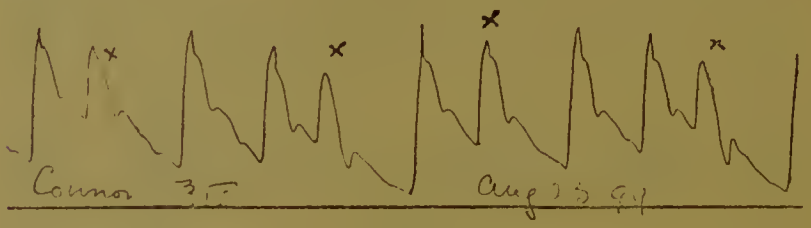

Fig. 7.

Shows a bigeminal and trigeminal form of pulse due to the occurrence of extra systoles, x, every second or third beat.

the pulse ceases to have any method in its madness, and is irregularly irregular.

We see, therefore, that no real distinction can be drawn between the intermittent and the irregular pulse. They are both in essence the same, and caused by the presence of these extra-systoles. The intermittent, the bigeminal, the trigeminal, and the irregular pulse are as a rule different degrees of the same condition.

The question we have now to ask ourselves is this—What is the cause of an extra-systole? Can nerve influences indirectly cause extra-systole?

There may be, as we have seen, the most varied groupings, and the irregularity may be so great that one might readily think that there was some serious cardiac lesion; but through it all the stimulus arising in the auricle continues to be pro-

duced with perfect regularity, though it is not always able to dominate the rhythm, because, in the commoner forms of extra-systole, when the normal stimulus reaches the ventricle it finds the latter either in systole or in the refractory period, and therefore unable to respond.

For the production of an extra-systole there must be direct stimulation of the heart muscle. It is neither impossible nor improbable that the power of automatic stimulus production rises so high in one or more portions of the heart-wall that an effective stimulus is occasionally generated at that point before the usual stimulus has arrived from the auricle.

The ætiological causes of these extra-systoles will be best discussed under the next head, namely, the class of patient in which these irregularities occur.

Extra-systoles may occur at any age; in health or in serious disease. They are found in the strong and in the weak. In fact, we cannot say that they are connected with any particular intrinsic or extrinsic conditions.

It is possible to classify the patients in whom these extra systoles occur into four groups. The following classification is that suggested by Wenckebach:—[1]

Group I.—In the first we find patients in whom the heart is irritable, or rather, in whom the cardiac musculature possesses an abnormally high excitability. These patients have no organic disease, they have a normal area of cardiac response, they live a healthy, vigorous life, and after death nothing abnormal can be detected in their cardiac muscle or valves. One can hardly believe that in such persons there are always pathological stimuli producing these extra-systoles, and it seems much more probable that there is an abnormally excitable heart muscle, either hereditary or acquired, so that sporadic stimuli, which would have no effect on ordinary individuals, are able to give rise in them to extra-systoles. In horses and dogs this excitable condition of the heart is found, and extra-systoles are of common occurrence.

[1] K. F. Wenckebach, *Arhythmia of the Heart*, p. 60.

This class must also be enlarged to include those who have what are called "irritable hearts." Their hearts, like their other organs, are easily stimulated into excessive activity, and as quickly exhausted. Here we are dealing with the large class of neurasthenics in whom the heart responds to much weaker stimuli than in those with strong and stable nervous systems.

Group II. includes patients in whom the extra-systole has no direct connection with the heart or the circulation, but in whom the extra-systole is the result of *reflex influences* or of *toxins*. Such conditions as constipation, worms, or a disordered stomach may be sufficient to originate extra-systoles. Cure the constipation, relieve the stomach, and the irregular action of the heart ceases. Again, toxic substances are a common exciting factor, and it is noteworthy that in the case of the toxins of the infectious fevers the extra-systoles are generally found during the convalescence. The toxins find the heart muscle exhausted and perhaps excitable after the strain of the fever. Tea, coffee, tobacco are also frequent causes of extra-systole.

In *Group III.* we have patients in whom there is some abnormal condition of the circulation, but no actual heart disease. It is still unsettled what part high blood-pressure plays in the causation of extra-systoles. We know that they may and do occur in patients with a raised blood-pressure, but this is not the rule. *Arterio-sclerosis*, however, is very often associated with extra-systoles, and it is probable that as the heart grows older its excitability is increased. Whether due to arterio-selerosis in the old, or simply age itself, it has long been noted that extra-systoles are very frequent in those of advanced years.

Group IV. comprises those who have undoubted organic disease of the heart. Broadbent states that the intermittent form of pulse is more frequent in the aortic cases, the irregular in mitral; but we know now that these two forms of irregular pulse are in essence the same.

It is easy to understand how dilatation, myocarditis, pericarditis, pericardial adhesions, and disease of the coronaries can all give rise to stimuli likely to initiate an extra-systole, but we cannot forget how many cases of serious organic disease of the heart go on to death with a regular and frequent pulse. These facts force home to us our ignorance as to the real cause of the extra-systole in such cases.

Up to this point we have attempted to obtain a clear idea as to what takes place in the heart during these forms of irregular action, and we have also roughly classified the patients who present this feature of extra-systoles.

Let us now consider the prognosis.

There can be no doubt that the tendency has been to take too grave a view of the condition of patients in whom there exists a cardiac arrhythmia. Most of the text-books refer to the subject in a vague and confusing manner, and when one meets with an arrhythmia, especially if it occurs in a nervous patient, one is liable to hedge and speak about a "weak heart," or even to take a grave and serious view of the patient's health.

As a rule, however, extra-systoles have little clinical importance, and afford only slight help in diagnosis and prognosis. Their presence, under certain conditions, may be of value in the prognosis; but when they are the only abnormal symptom present, or when they are associated with the milder degrees of heart failure, then they suggest none of those consequences so gravely hinted at by some.

In the heart of the aged an irregular pulse is in itself of no moment. When extra-systoles are habitually present, the advent of a frequent pulse tends to cause them to disappear. This is often noticed with the frequent pulse of pyrexia.

If, however, in a patient with a high temperature extra-systoles are noted for the first time, or if they persist in spite of the increased frequency of the heart's action, then we are justified in concluding that there is a severe toxæmia.

Extra-systoles occur frequently during convalescence: they then signify nothing beyond the existence of a weak

and excitable heart. The pulse is infrequent, the diastolic period is long, and consequently the opportunities for abnormal stimulation to excite a response are increased.

In the acute stage of pneumonia or rheumatic fever, in which the pulse is frequent, the early appearance of extra-systoles bodes ill for the patient. A pneumonia patient with a pulse which intermits during the first five days of the attack rarely recovers, but irregularities at the crisis or during convalescence matter little. This rule holds in the majority of instances; but it cannot be considered absolute, as six cases at least have come under my notice where there was an irregular pulse present before the crisis, and where the patient recovered from his pneumonia. They are referred to in more detail in my paper on pneumonia in the *Lancet* of June 11th, 1904.[1] [2]

In estimating the prognosis in the absence of fever, Broadbent suggests that the patient should walk smartly for a minute or so, and, he says, if the intermittency disappears the prognosis is much happier than if the irregularity becomes more marked. The rationale of this procedure is obvious, when one remembers that exercise increases the frequency of the heart's action, and so diminishes the periods of diastole during which the heart can respond to extra stimuli.

In any heart case, after forming an opinion as to the pathological condition present, if there be one, the outstanding factor of importance in the prognosis is the power of the heart to do its work—that is, its "area of response." We are far too much influenced by murmurs, thrills, and sounds. When we have no reason to believe that the pathological condition is serious, virulent, or progressive, and when we find that the "area of cardiac response" is good, the presence or absence of extra-systoles is immaterial. In forming an opinion as to the prognosis in any given case, the occurrence of extra-systoles is generally the least important fact to be considered.

[1] "Acute Lobar Pneumonia, Two Hundred Cases of," *Lancet*, June 11th, 1904.
[2] *The Pulse*: J. Mackenzie.

It is, however, most necessary to bear in mind that irregular action of the heart is sometimes brought about by causes other than extra-systoles, and it is most important both for diagnosis and treatment that one should be able to state with certainty in any given case whether the irregularity is due to extra-systole or not. For example, an intermittent pulse may be caused by depression of conductivity. In such a case the intermission may be regular or almost regular (as in fig. 8), or irregular (as in fig. 9); but a careful analysis of the venous tracings in these cases reveals that, when the irregularity occurs, there have been two auricular contractions to one

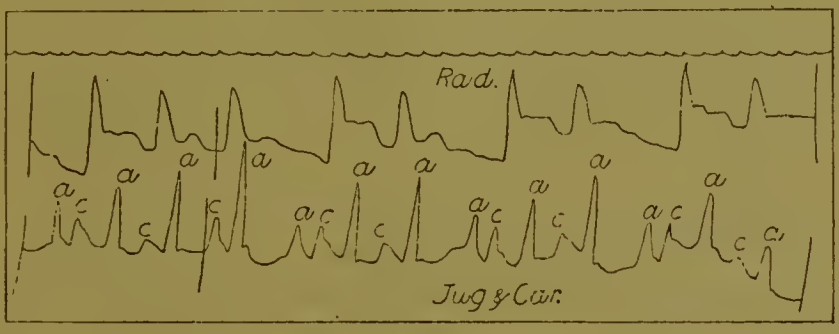

FIG. 8.

Shows the same as fig. 9, except that there is a tendency to rhythmical irregularity.

ventricular.[1] [2] The ventricle, instead of initiating its own stimulus, as in the case of an extra-systole, fails to receive its normal stimulus from the auricle, because of lowered conductivity in the muscle fibres connecting the auricles with the ventricles—the bundle of His. In this case a ventricular silence occurs at the time of the irregularity, and we do not hear the odd, tumbling sounds which can be made out during an extra-systole.

Arrhythmia, due to depression of conductivity, may be brought about in certain instances by the administration of

[1] See also *Brit. Med. Jour.*, Oct. 21st, 1905, "Pathology of Bradycardia"; and *Lancet*, Jan. 20th, 1906, "Bradycardia and Cardiac Arrhythmia."

[2] "New Methods of studying Affections of the Heart," J. Mackenzie, *Brit. Med. Jour.*, March and April 1905.

digitalis,[1] or it may follow an attack of influenza. Many cases, however, occur where the causal agent can not be traced.

There is another group of irregular heart action which should be differentiated from that of extra-systole. I refer to those hearts in which there is no longer the rhythmical production of a physiological stimulus in the auricle—in which, therefore, the auricle has ceased to dominate the rhythm. Here the dominating stimulus does not arise in the auricle, but in or near the muscle fibres joining the auricles and ventricles, and which, as Gaskell has pointed out, have a highly developed power of stimulus production. The stimulus arising at this

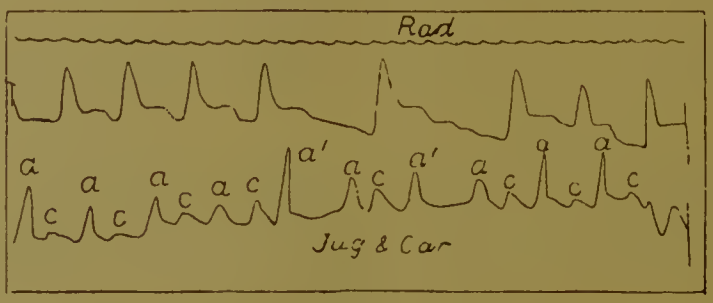

Fig 9.

"Tracings of the radial taken the same time as the jugular and carotid. The waves *a* and *a'* are due to the auricle, and appear with perfect regularity, while the radial pulse and carotid (wave *c*) appear irregularly, a beat missing after each auricular wave *a'*."

point passes simultaneously up into the auricle and down into the ventricle, and as a result the auricle and ventricle are in systole at the same moment.

This condition is found in many patients said to be suffering from myocardial degeneration, and also in the later stages of mitral stenosis. Figs. 10 and 11 are traeings taken from such patients in whom the pulse was markedly irregular. An analysis of the venous pulse traeing shows that the auricle did not contract before the ventricle. This form of arrhythmia tends to be permanent, and is always associated with limitation of the area of cardiac response. In the *British Medical Journal*

[1] J. Mackenzie, *Brit. Med. Jour.*, March and April 1905.

of October 21st, 1905, I have discussed this form of arrhythmia at length.

Fig. 12 is a good example of yet another form of arrhythmia due to a cause other than extra-systole. Here the dropped

Fig. 10.

The tracing taken from the jugular vein shows the ventricular type of venous pulse. There is no sign of any contraction of the auricle preceding the period of ventricular systole, E. The heart was beating infrequently, 40 to 45 beats per minute. The patient, a man of 41, suffers from mitral stenosis and regurgitation, and there is very marked diminution in his "area of cardiac response."

beat at A and the small beats at B and C are the result of impaired contractility. That this is not the result of extra-systole is obvious on carefully examining the tracing and comparing it with figs. 3, 5, 6, and 7.

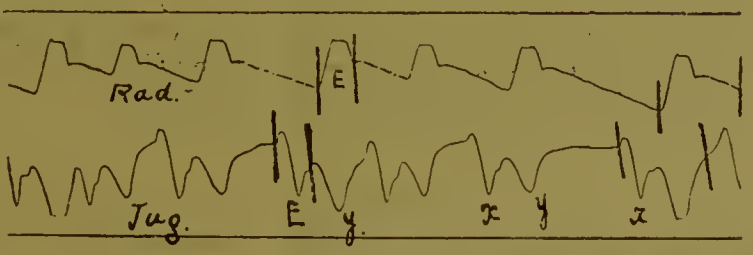

Fig. 11.

The tracing taken from the jugular vein shows the ventricular type of venous pulse. Here there is no rise in the tracing corresponding with *a* in the first three tracings; the auricle has not contracted before the systole of the ventricles. The patient, a man aged 65, suffers from slight mitral incompetence, and there is marked diminution in his "area of cardiac response." Two months ago he was at work.

In every case of ventricular extra-systole the extra-systole or premature beat occurs earlier than is normal: in fig. 12, on the contrary, the small beat at B and C occurs at the very moment when it should normally appear. Again, in

extra-systole there is a compensatory pause following on the extra-systole, so that a longer time elapses between the extra-systole and the subsequent ventricular systole. There is nothing of the kind in this tracing: the time between the smaller pulse-waves and the next pulse-wave is the same as between any two normal systoles.

Sometimes this variation becomes rhythmical, and the heart gives alternate strong and weak contractions; the pulse is then named the "pulsus alternans."[1] [2] The cause of this condition is now admitted to be failure in contractility of

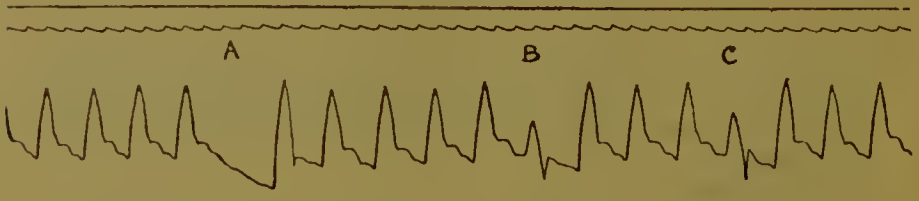

Fig. 12.

Irregular pulse caused by depression of contractility. Tracing obtained from a patient suffering from a septic throat. There was marked constitutional disturbance.

Note that at A the ventricular systole quite fails to transmit a wave to the wrist; at B and C the wave at the wrist is small and the systole of the left ventricle is therefore much weaker than those preceding and following it.

Note that there is no compensatory pause following on the small wave, and that the small wave is equidistant from those before and after.

the cardiac muscle. An alternating pulse is regarded in a serious light when it occurs in the course of acute febrile diseases, such as pneumonia.

I wish here to emphasise the great importance of differentiating between the ordinary extra-systole and other irregularities such as I have exemplified, and to point out that in many cases the recognition of these various irregularities can only be satisfactorily determined by an analysis of the venous tracing.

[1] Wenckebach, *Arhythmia of the Heart*, p. 109.

[2] J. Mackenzie, "The Action of Digitalis on the Human Heart," *Brit. Med. Jour.*, March and April 1905.

Treatment.—It is obvious from what has gone before that there can be no uniform line of treatment. Where treatment is considered advisable, it must be based on the etiology and on the general condition of the patient. All reflex causes, such as dyspepsia, flatulence, constipation, helminthiasis, should meet with appropriate treatment. If considered necessary, tea, coffee, and tobacco should be prohibited. Gout should be carefully treated.

The irritable and excitable heart muscle of neurasthenia should be braced up by regulated exercise and rest, and the fears of the patient as to the existence of serious cardiac mischief assuaged. Among drugs, sodium bicarbonate, bromides, dilute hydrobromic acid, digitalis, belladonna, camphor monobromate, and general tonics all find a place.

Summary.—In conclusion, we see that many of the old classifications of cardiac arrhythmia are meaningless and superficial, suggesting differences which do not exist. In most cases the intermittent pulse, the bigeminal, the trigeminal, and irregular pulse are all due to the same variation from the normal method of systole—namely, the extra-systole. This extra-systole is the response of the ventricle to a stimulus arising in itself, and antecedent in time to the arrival of the normal stimulus travelling from the auricle. The result of such an abnormal contraction is a break in the regular normal pulse-beat. As a rule the presence of extra-systoles is of little moment. When, however, they fail to disappear with an increased frequency of heart-action, or when they appear during pyrexia, as in pneumonia or acute rheumatism, the outlook is grave.

An analysis of simultaneous arterial and venous pulse tracings enables us to distinguish with certainty between extra-systoles and irregularities due to other and probably more serious conditions.

I am much indebted to Dr Mackenzie for permission to publish tracings 2, 6, 8, and 9.

STOKES-ADAMS DISEASE. REPORT OF A CASE.

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In the large majority of the cases of Stokes-Adams disease bradycardia is a marked and persistent symptom. In these cases the syncopal and convulsive seizures are associated with exacerbations of the bradycardia, during which the ventricles may remain in diastole for periods of one or more minutes. Such cases are chronic, and may persist for many years. I have at present one patient of this type under observation who has suffered from a most infrequent pulse for nine years, complicated latterly by convulsive attacks; another for over three years. As an example of an extreme prolongation of this condition, Osler [1] records a case of a man whose pulse was infrequent, and who suffered from fainting attacks for thirty years; he finally died in one of these attacks.

There is another and much less frequently observed type, in which the pulse-frequency is normal except during the seizures, which are paroxysmal and transient. These cases are sometimes acute in character and rapidly fatal. Such is the type of case I am about to describe.[2]

The patient, M. P., a woman aged 40, was admitted on March 26th, 1904, into the Stanley Hospital, under the care of my colleague, Dr Whitford, and I am indebted to him for permission to publish the case. When admitted, the patient was somewhat emaciated, and suffered from extreme anæmia. Her temperature for the first three weeks was irregular; the pulse-frequency varied from 96 to 108. On admission, there was a faint trace of albumen, which disappeared at the end of a week, and was not noted again until a temporary reappearance on the 2nd July: urine otherwise normal.

[1] W. Osler, "On the so-called Stokes-Adams Disease," *Lancet*, Aug. 22nd, 1903.

[2] G. A. Gibson, "Bradycardia," *Ed. Med. Jour.*, July 1905.

On examination, the thoracic and abdominal viscera were apparently normal; auscultation of the heart, however, revealed hæmic bruits. Examination of the blood gave 1,330,000 red corpuscles per cub. mm. on May 7th; but microscopical examination did not show the typical picture of pernicious anæmia. On June 8th the number of red corpuscles was 1,650,000 per cub. mm. During this period she was treated with increasing doses of arsenic, with not altogether satisfactory results. On June 27th, at 11 a.m., the patient entered on a new phase. On this morning at 11 and 11.45 a.m. she had two seizures which were thought to be epileptic in nature.

The second fit was described as "heavy." At 5 p.m. there was another severe attack, during which the breathing ceased, and artificial respiration was performed. On the 28th, fits occurred at 2 a.m. and 5 a.m.; two fits are noted at 8 a.m.; at 11 a.m. Dr Sorapure, one of the house-physicians, was called, and later he made the following note: "She was convulsed, body and limbs rigid and moving slightly, head thrown back and the neck muscles standing out; the lower jaw moving with respiration, which was slow, forced, and shallow; the face was livid. This condition subsided in about a minute; she lay quite still, became very pale, and the pulse could not be felt at the wrist. A feeble pulse became perceptible, and she suddenly regained consciousness. The head was lowered, and saline enemata were given. Twenty minutes later she had a similar attack; but on this occasion the respiration ceased entirely, and the face became quite livid. The pulse stopped; artificial respiration was resorted to, and was continued for five minutes, when a few shallow gasps indicated returning activity of the centre. Strychnine was now administered, but the examination of the pulse was neglected during the more urgently needed efforts. In fifteen minutes she had another convulsion similar to the first—the respiration not being suspended." At 2 p.m. and at 4.25 p.m. there were slight attacks. Attacks were also noted at 4.30, 4.40, 4.45, also at 9, 9.5, 9.30, and 9.35 p.m. In the absence of Dr Whitford, I was

asked to see the patient, whose condition was considered urgent. I saw her just after the attack at 4.25. She talked rationally, and then complained of a sinking feeling and sensation of faintness. This was the precursor of the attack. There was a slight convulsion, some congestion of the face, during which neither pulse nor heart-beat could be made out. Associated with these symptoms there was loss of consciousness. She regained consciousness for a few seconds only, then became pale and relapsed into coma. Her flaccid lips blew out and in, her heart stopped (this fact was ascertained by auscultation), and for ten seconds no pulse could be felt at the wrist. The pallor of the face was then replaced by a more normal colour, and simultaneously she became rigid and arched her back. The return to consciousness was immediate and sudden; she recognised those about her and spoke to them. The pulse was perceptible just before consciousness returned. It was noted that on several occasions the return to consciousness was followed by retching, which lasted for a few minutes. Another attack was initiated by nothing more than pallor and a feeling of faintness, followed by a sudden loss of consciousness. The conjunctival reflex was present throughout; the pupils were a little dilated.

After having watched the patient in these seizures and heard an account of the previous attacks, I had no doubt that we were dealing with a condition of Stokes-Adams disease.

As a rule the knee-jerks were increased, and there was flaccidity during the attacks until just preceding the regain of consciousness, when the limbs became rigid, and the patient was possibly convulsed. In the more severe fits there was involuntary passage of urine and fæces.

I auscultated the chest during several fits. When the breathing was stertorous, the stertor masked all other sounds; but when the breathing ceased or was very shallow no heart-sounds were audible. Before the return of consciousness, however, very faint sounds were heard, rapidly increasing to loud thumping beats slightly irregular in time. Sometimes

the first sound was reduplicated. The patient generally recovered consciousness a moment or so subsequent to the first faint heart-beats. The pulse-rate between the attacks was 100 to 115, regular in force and frequency, of poor volume and low tension. Systolic bruits were heard about the base of the heart, and over the tricuspid area. The temperature rose to 100° F. on the evening of June 28th.

On the 12th July I made the following note: "Since the 28th of June the patient has been free from fits; there has been no loss of consciousness; the pulse has been continuously between 84 and 128, averaging about 100 per min. Up to the 8th July she has been fed entirely by nutrient enemata. This was necessary because she could retain no food. On Friday the 8th feeding by the mouth was started. One and a half pints of fluid were administered in the twenty-four hours, a pint of which was retained. The tendency to vomit is now much less than it was. The tongue is very dry, the blood-pressure is low, there is very marked insomnia, and more or less constant frontal headache. She is rather dazed and fretful, and her general condition most unsatisfactory." On the 16th July the temperature ran up to 103° F., pulse-frequency 120 to 140 per min. Her condition became steadily worse, and she died on the 20th July.

A post-mortem examination was made on July 22nd. The body was wasted, skin yellowish; there was slight œdema of the ankles. There were old adhesions over both lungs. The left lower lobe was solid, and sank in water. The apex of the left lung showed signs of old tubercle. The heart was small, pale, and fatty, and weighed 8 oz.; the muscle substance very friable. There was no general arterio-selerosis, and the coronary arteries were normal. The liver was large, pale, and fatty, and weighed 58 ozs. The stomach was small and thin-walled, with marked ecchymosis of the fundus. The marrow was normal. There were no signs of nephritis. It is to be regretted that no examination of the central nervous system was made.

Remarks.—The outstanding feature of importance and interest in this patient is the repeated occurrence of sudden and complete loss of consciousness, accompanied by cessation of the heart's activity, and in the more severe seizures cessation of respiration. Of such attacks there were nineteen: the first occurred at 11 a.m., June 27th, the last at 9.30 p.m., June 28th. There were all grades of severity, from a short attack lasting a few seconds, initiated by a feeling of faintness, and characterised by pallor, cardiac arrest, sudden loss of consciousness, yet without convulsive movements or rigidity, to a severe attack in which the patient was looked upon as dead, all the usual signs of life being absent, and breathing only returning after artificial respiration had been performed for about five minutes. It is obvious from the description of the fits that they were not epileptic. The slighter seizures had much more the appearance of syncope, while the severer fits were similar to the seizures noted in those cases of bradycardia in which occur paroxysms of still greater reduction in the heart-frequency.

In the last eighteen months I have had under observation a patient suffering from marked bradycardia.[1] During the last five months there have been six periods during which he has been liable to "fits." The following is a description of one of the more severe seizures, and on comparing it with those in the case of Mrs M. P. it is evident that they are similar in nature.

In this man the fit began abruptly with loss of consciousness, his head fell back, he gave a few deep stertorous respirations, his face became ashen grey, breathing then ceased, he shortly became rigid, some convulsive twitching of the hands and arms occurred, his face flushed, and simultaneously the pulse, which had been absent from the beginning, was felt at the wrist, smaller than usual, and rapidly increasing in volume. With the return of colour he sighed deeply, woke up suddenly, and looked around. In this case it has been demonstrated beyond doubt that there is a condition of heart-block. The auricles

[1] "Bradycardia and Cardiac Arrhythmia," *Lancet*, Jan. 20th, 1906.

have a different frequency from the ventricles, and during the seizures continue to contract, although the ventricles are quiescent. I think there can be no doubt that in the case of Mrs M. P. we are dealing with one of those rarer cases of paroxysmal heart-block in which the pulse is frequent between the attacks. In the case of the man referred to above it is nearly always possible to demonstrate some depression of conductivity as a cause of his bradycardia, whereas in Mrs M. P. the average pulse-frequency was 90—showing that between the attacks there was probably no serious impairment of this function.

Dr Handford published in the *British Medical Journal* of December 31st, 1904,[1] a fatal case of heart-block due to the presence of gummata in the auriculo-ventricular groove, which obviously interfered with the normal conduction of the stimulus from auricles to ventricles. The symptoms noted "consisted in great disturbance of the cardiac rhythm, with frequent periods of apparent cessation of the heart's action lasting for five or fifteen seconds or longer. There was no pulse at the wrist and no cardiac impulse. The face became pale; there was a slight general convulsion from cerebral anæmia; then a few deep respirations were followed by a resumption of the regular heart's action."

Concerning the pathology of heart-block, one factor stands out with prominence—the presence of a diminution or loss of conductivity in the muscular connection between the auricles and the ventricles: the auriculo-ventricular bundle of His—"the pathway of the stimulus to contraction."

As a consequence of this, the auricle and ventricle may beat independently, each with its own rhythm, or the ventricle may only respond to every second, third, or even fourth beat of the auricle. This alteration of function may be due to the effect of *toxins*, and explains the bradycardia after such diseases as influenza, typhoid, diphtheria, etc.—the "post-febrile group"

[1] H. Handford, "Remarks on a Case of Gummata of the Heart," *Brit. Med. Jour.*, Dec. 31st, 1904.

in Osler's classification of Stokes-Adams Disease. Osler[1] records such a case following a streptococcal pharyngitis, and Mackenzie[2] one as a result of influenza.

The depression of conductivity may also be caused by *organic changes* in or near the bundle of His: as in the case described by Stengel,[3] where there was a "patch of atheromatous character, sclerotic and white," extending "to the endocardium exactly over the bundle of His, where this band passes from the ventricle to the auricle."

Handford's case already mentioned also illustrates this point, and in a case I reported in the *British Medical Journal* of October 21st, 1905,[4] Keith—to whom I sent the heart—described a "stretching of the auricular canal and attenuation of the bond between the bases of the valves (tricuspid) and base of the ventricle." This would necessarily involve the bundle of His.

It is well known what extensive myocardial degeneration there may be as the result of severe sclerosis of the coronary arteries, and it is a remarkable fact that the large majority of patients suffering from Stokes-Adams disease have arteriosclerosis in an advanced degree.

In the reports of 41[5] autopsies collected from various sources, it was noted that 33 patients showed definite and often marked sclerosis of the coronary arteries. In two of these 33 patients the myocardium was normal. In 32 of the 41 patients there was definite myocardial degeneration.

I made a post-mortem recently on such a case, and have included it in the above statistics. The coronaries were rigid

[1] W. Osler, "On the so-called Stokes-Adams Disease," *Lancet*, Aug. 22nd, 1903.

[2] J. Mackenzie, "New Methods of studying Affections of the Heart," *Brit. Med. Jour.*, March, April 1905.

[3] Stengel, "A Fatal Case of Stokes-Adams Disease, with Autopsy showing Involvement of the Auriculo-Ventricular Bundle of His," *Amer. Jour. Med. Scien.*, Dec. 1905.

[4] "On the Pathology of Bradycardia," *Brit. Med. Jour.*, Oct. 21st, 1905.

[5] R. T. Edes, *Philadelphia Med. Jour.*, 1901.; J. E. Grieve, "The Etiology of Bradycardia," *New York Med. Jour.* and *Phil. Med. Jour.*, July 1st, 1905; A. Webster, "Cardiac Arrhythmia in relation to Cerebral Anæmia and Epileptiform Crises," *Glasgow Hosp. Rep.*, 1901.

and calcified, and the right almost completely occluded, the occlusion being of old standing.

In Mrs M. P. (also included) the coronaries were normal, but the heart showed very advanced fatty degeneration, to be accounted for by the extreme and prolonged anæmia. In the presence of such general myocardial degeneration it seems reasonable to presume that the bundle of His did not escape, and in consequence its function was impaired.

Erlanger, who has published a most valuable [1] account of his work on the auriculo-ventricular bundle of the dog, holds that all the cardinal symptoms of Stokes-Adams disease may be duplicated by heart-block resulting from a lesion in or near the bundle of His, and by this alone. He also states that no typical case of Stokes-Adams disease has been described in which a heart-block might not have been the cause of the trouble. He seems to ignore stimulation of the vagus as a possible factor in causing or determining these peculiar seizures.

We know that stimulation of the vagus causes depression of all the functions of the heart,[2] and possibly, in the presence of other factors, stimulation of the vagus might on occasion depress one function more than others.

I had the pleasure of seeing with Dr Mackenzie a patient in whom this fact was demonstrated to perfection.

The patient—a man—had slight valvular disease, and with it some depression of conductivity. Whenever he swallowed, an arrhythmia was produced, one or two ventricular beats dropped out, and then the heart regained its usual steady rhythm. On analysing the venous pulse one found that the stimulation of the vagus caused by swallowing had greatly increased the depression of conductivity, and so brought about temporary heart-block. The venous pulse also demonstrated that the auricles had continued their rhythm unchanged.

[1] J. Erlanger, "On the Physiology of Heart-block in Mammals, with special reference to the Causation of Stokes-Adams Disease," *The Journal of Experimental Medicine*, vol. vii. No. 6, vol. viii. No. 1.

[2] Gaskell, *Schafer's Physiology*, vol. ii.

Chauveau [1] obtained a similar phenomenon by experimentally stimulating the vagus in a dog; the ventricular systoles dropped out as a result of the depression of conductivity.

When considering the question of vagus influence, it is interesting to recall that in Mrs M. P. the period of ventricular silence ended with the appearance of faint feeble cardiac contractions which rapidly increased in force. Laslett [2] also observed this termination in a patient, and Webster [3] published beautiful tracings of a similar condition. Concerning Webster's case, Wenckebach [4] says: "Anyone who has stimulated the vagus in experiments will at once observe the effect of vagus stimulation in this tracing." He considers the gradual increase in size in the pulse-waves as an example of "Bowditch's staircase," and states that Engelmann has demonstrated that there is here a diminution of contractility. In his opinion, everyone will be persuaded that Webster's case is one of arrest of the heart from the action of the vagus, where the negative inotropic influence (depression of contractility) is the most prominent symptom.

The physiology of vomiting is complicated, and it is suggestive that the vomiting centre in the medulla, like that of the respiratory centre, is intimately related to the central connections of the afferent and efferent fibres of the vagus. The fact that the attacks in Mrs M. P. were followed in the majority of instances by vomiting, and that vomiting was a persistent symptom for some days after the 28th June, is of considerable interest, and lends some support to the view that the vagus is not a negligible factor.

It must not be forgotten, however, that both the apnœa during the attack and the vomiting following on the attack

[1] Chauveau, "De la dissociation der rhythme auriculaire et der rhythme ventriculaire," *Rev. de Méd.*, Paris, 1885.

[2] E. E. Laslett, "A Case exhibiting the Adams-Stokes Syndrome," *Lancet*, June 4th, 1904.

[3] A. Webster, "Cardiac Arrhythmia in relation to Cerebral Anæmia and Epileptiform Crises," *Glasgow Hosp. Rep.*, 1901.

[4] K. F. Wenckebach, *Arhythmia of the Heart.*

may have been the result of the changes in the bulb caused by the cardiac arrest, so characteristic of the seizures.

Erlanger demonstrated, in the work already referred to, one most important fact, namely, that when heart-block was complete, stimulations of the vagus failed entirely to affect the frequency of the ventricle, so that in those cases of Stokes-Adams disease in which the heart-block is complete, some other explanation must be found than that of vagus intervention, and the cause will probably be discovered in the heart itself.

On the other hand, in those cases where the heart-block is not complete, as in Mrs M. P. and in Handford's case, may not stimulation of the vagus be sufficient to produce either sudden and complete block or heart "stand-still" by direct action on the ventricle? In my opinion, Erlanger seems unduly to minimise the effect of vagus stimulation as a possible determining factor in certain instances of Stokes-Adams disease.

medical student is imperfectly instructed in these ailments, lock hospitals in our large centres are few and far between, and the British soldier and sailor, the most expensive articles of their kind in Europe, are protected by the latest advances in sanitary science from enteric and cholera, but allowed every opportunity of acquiring syphilis and other venereal diseases. In relation to this I quote :—

"A contagious disease is weakening our army. In the case of any other infectious disease, such as cholera or enteric fever, elaborate measures are taken and carried out with success. Syphilis, being of venereal origin, is put by the powers that be upon a totally different footing. If the State expects the army to protect the country, it is the duty of the State to protect the army against disease of any kind, whether of venereal origin or not."

The condition of England, in civil and military life, as regards syphilis is in my opinion a great and growing danger.

On the continent of Europe, especially in Germany, things are on a different footing, no consider[illegible]